ASDS
American Society for Dermatologic Surgery
American Society for Dermatologic Surgery Association

March 1, 2010

The Honorable Jonathan A. Harris
The Honorable Elizabeth B. Ritter
Co-chairs, Public Health Committee
Connecticut State Legislature
Room 3000, Legislative Office Building
Hartford, CT 06106

Re: SB 263

Dear Senator Harris and Representative Ritter:

As President of the American Society for Dermatologic Surgery Association (ASDSA), a medical specialty organization representing over 5,200 physician members across the nation, I am writing to urge the Public Health Committee to support SB 263, to require children under the age of 16 to obtain with written parental consent prior to indoor tanning, and to require indoor tanning facilities to post signs notifying customers of the health risks associated with indoor tanning.

As skin cancer surgeons, members of the ASDSA see first-hand the detrimental effects of early exposure to ultraviolet rays. **According 2009 survey, our members performed over 3.1 million skin cancers procedures last year, which is a fifty-five percent increase over 2005.**

Last year, the International Agency for Research on Cancer, the cancer division of the World Health Organization, classified tanning beds as "carcinogenic to humans" — the agency's highest cancer-risk category, which also includes radon gas, plutonium and radium.

**Total doses of ultraviolet rays from a tanning bed may be as much as five times more than natural sunlight. This means that 20 minutes spent in a tanning salon may be equal to 2-3 hours in the noontime sun**, according to the attached scientific article, "Tanning and Cutaneous Malignancy" (Ibrahim, S; Brown, M; *Dermatol Surg* 2008;34:460–474). The article states further,

> "The World Health Organization has estimated that in the year 2000, up to 71,000 deaths worldwide were attributable to excessive UV exposure. In the United States, skin cancer accounts for more than 50% of all malignancies, with well over 1 million cases of nonmelanoma skin cancer and 60,000 cases of malignant melanoma estimated for 2007 (melanoma in situ will account for an additional 46,000 cases). Mortality from melanoma in the US occurs at the rate of approximately one life per hour or more than 8,000 deaths annually."

For these reasons, ASDSA believes that stronger safeguards should be in place to protect children from unnecessary exposure to the ultraviolet rays in indoor tanning salons, such as those proposed in SB 263.

Thank you again. Should you have any questions please do not hesitate to contact Director of Advocacy and Public Policy Lisle Poulsen at (847) 956-9126 or *lpoulsen@asds.net*.

Sincerely,

ASDS
American Society for Dermatologic Surgery
American Society for Dermatologic Surgery Association

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# Tanning and Cutaneous Malignancy

SHERRIF F. IBRAHIM, MD, PHD, AND MARC D. BROWN, MD*

BACKGROUND Exposure to ultraviolet radiation (UVR) results in a darkening of the skin known as tanning. Recently, it has been shown that tanning is a response to UVR-induced DNA damage and represents the skin's efforts to protect itself against further injury. Despite the link between UVR and cutaneous malignancy, people continue to pursue tanning from natural and artificial sources. This trend is reflected in the exponential rise in skin cancer incidence.

OBJECTIVE The objective of this study was to review our current understanding of the factors controlling the tanning response and the relationship to cutaneous carcinogenesis, as well as the impact that the multibillion dollar tanning industry has had on the practice of dermatology.

MATERIALS AND METHODS Extensive literature review was conducted in subjects related to tanning and the relationship to cutaneous malignancy.

RESULTS Our knowledge of tanning and its effects on the skin has increased tremendously. It is clear that tanning contributes to the development of skin cancer. Despite this information, the incidence of skin cancer continues to increase exponentially.

CONCLUSIONS Skin cancer poses a major public health concern and tanning remains the most modifiable risk factor in its etiology. Social, economic, and legislative issues have become tightly intertwined with the complex nature of human behavior in the continued pursuit of an activity that clearly has detrimental effects on one's health.

*The authors have indicated no significant interest with commercial supporters.*

## The History of Tanning

Every great civilization has had an unwavering reverence for the sun. Beyond a central role it has played in religion and mythology, the sun has also been inextricably tied to the practice of medicine. Sunlight was used by the ancient Egyptians to treat vitiligo and by the Greeks to restore good health to the ill.[1] By the end of the 19th century, the connection between sunlight and rickets was made;[2] and soon after, the earliest treatments resembling modern phototherapy emerged, primarily as a remedy for lupus vulgaris.[3] The years that followed saw an abundance of proponents for increased sun exposure, with prominent doctors at the time confidently asserting phrases such as "sunlight to the skin protects against development of cancer in parts of the body that are less accessible for treatment,"[4] and that for infants, "the sunbath is just as important as the water bath."[5] From a socioeconomic view, however, tan skin was an indicator of outdoor hard labor and low class, while "porcelain paleness" was the epitome of high society.[1] With the arrival of the Industrial Revolution, there was a shift of the labor force from the fields to the factories, eventually leading to a reversal of the association between skin color and class implication. With time, a deep, dark tan came to symbolize wealth, free time, and well-being. An interview with the 1920s fashion icon Coco Chanel is credited with single-handedly sparking the tanning craze across the Western World after she was photographed on the Duke of Westminster's yacht proclaiming that "the 1929 girl must be tanned . . . A golden tan is the index of chic."[6] This sent flocks of women to the beach in pursuit of the perfect tan: with the introduction of the bikini in 1946, further loosening of the moral restrictions on exposing one's skin in the 1960s, and the burgeoning

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movie industry in Southern California, modern tanning culture was here to stay.

The data linking ultraviolet radiation (UVR) and skin cancer were not far behind. As early as 1907, Unna[7] noted that grape pickers in France developed increased numbers of skin cancers on sun-exposed areas. Experiments by Findlay[8] and Roffo[9] were among the first to demonstrate that rodents exposed to UVR developed skin cancer, and by the second half of the 20th century, there was overwhelming evidence implicating UVR as a carcinogen. Significant contribution to understanding the mechanisms by which these cancers developed was made by Cleaver in 1968,[10] when deficient DNA repair was demonstrated in cells from xeroderma pigmentosum patients.

Over the past 40 years, the association between sun exposure, prematurely aged skin, and cutaneous malignancy has become indisputable; however, the desire to tan is arguably higher than ever before. In a survey of 8,000 Americans, 94% of respondents were concerned that exposure to ultraviolet light could lead to skin cancer, yet 68% also felt that they look better and healthier with a tan.[1] This increasingly common view has contributed to the enormous success of the commercial tanning industry. The first advertisement for a sunlamp appeared in *Vogue* magazine in 1923, but it was not until 55 years later that the first commercial tanning center in the United States opened in Arkansas.[1] Over the next 10 years, close to 20,000 tanning facilities had opened for business, with the current estimate lying somewhere between 30,000 and 50,000.[11,12] While there are well-established applications for phototherapy in dermatology, there is little sound evidence for any medical benefit afforded by recreational tanning. We are currently experiencing a skin cancer epidemic, and exposure to UVR remains the single most modifiable risk factor for the prevention of cutaneous malignancy. Discoveries in the laboratory as well as clinical and epidemiologic studies have supported this notion and firmly linked UVR with the development of skin cancer. The remainder of this article will highlight some of the more recent contributions from basic science that have led to a deeper understanding of the factors controlling the tanning response and the events leading to cutaneous carcinogenesis. The discussion will then review the impact that the multibillion dollar commercial tanning industry has had on the practice of dermatology. Along these lines, social, economic, and legislative issues have become tightly intertwined with the complex nature of human behavior in the continued pursuit of an activity that clearly has detrimental effects on one's health and is a major public health concern.

## The Basics of Tanning Science

While the earliest hints that solar radiation was involved with the development of skin cancer were based solely on clinical observation, our current understanding of physiologic tanning has been revealed with exquisite detail at the molecular and cellular levels. The intrinsic color of one's skin is determined by the content of pigment (melanin) within the basal layer of epidermal cells. While the number of melanocytes is relatively constant from one person to another, the variations seen across ethnicities is largely determined by the number and size of melanosomes distributed by the melanocytes to neighboring keratinocytes. Fitzpatrick categorized these differences into six skin types according to an individual's ability to tan and their proclivity toward sunburn.[13] This classification has remained as one of the most accurate means of risk stratification for the development of cutaneous malignancy.

Ultraviolet light is subdivided into three sections based on wavelength: UVA is composed of photons from 320 to 400 nm, UVB from 280 to 320 nm, and UVC from 200 to 280 nm. UVC and the majority of UVB are effectively filtered by ozone ($O_3$) in the Earth's atmosphere, such that the ultraviolet light incident upon the Earth's surface is composed of 95% UVA and 5% UVB. The effects of UVR on the skin include inflammation, erythema, sunburn,

immunosuppression, photoaging, cancer, and pigmentation (tanning). The tanning response is biphasic, composed of immediate pigment darkening (IPD), and delayed tanning. In darker skinned individuals, IPD occurs within seconds of exposure to UVR and does not involve new melanin synthesis, but oxidation of preexisting melanin and a redistribution of melanosomes to a peripheral dendritic location.[14] It is minimally photoprotective. Delayed tanning, on the other hand, refers to de novo melanogenesis. There is an increase in melanocytic activity and proliferation and a thickening of the stratum corneum and epidermis. This becomes evident 3 to 5 days after exposure to UVR and acts to protect the cell's genetic content from further damage by the formation of a nuclear cap of melanin that shields the nucleus.[15]

UVB is referred to as a complete carcinogen because its energy is directly absorbed by DNA and causes crosslinking of adjacent DNA base pairs. The resultant photoproducts are known as cyclobutane pyrimidine dimers and pyrimidine 6-4-pyrimidones and are unique to UVB irradiation. Their presence creates a bulky steric hindrance that disrupts replication and transcription of DNA and thus becomes genotoxic. The so-called UVB signature or fingerprint is a result of this process and manifests as C→T and CC→TT mutations in genomic DNA. Because of these direct effects on DNA, UVB photons are largely responsible for sunburn, tanning, and carcinogenesis.[15]

UVA, on the other hand, is not absorbed directly by DNA, but by other chromophores in the skin. As these chromophores are excited, reactive intermediates are generated, including oxygen free radicals, which are then capable of causing single- and double-stranded DNA breaks. Although UVA is 2 to 3 orders of magnitude less efficient than UVB in inducing the above changes,[16] there is strong evidence supporting significant contribution by UVA to oncogenesis.[17,18]

### Tanning Is a Response to DNA Damage

Once injury to DNA is detected, various DNA damage response pathways are invoked and the tanning response is initiated. It has been shown that patients who develop a basal cell carcinoma (BCC) before the age of 50 have significantly lower DNA repair capacity compared with matched controls with similar sun exposure and complexion, as well as an overall reduction in repair capacity in all patients with age.[19]

Central to the process of tanning is the role of p53, commonly regarded to as the "Gatekeeper of the Genome." It is a tumor suppressor protein that acts as a transcription factor for numerous other genes as a response to cellular stressors (including UVR) by preventing the reproduction of cells with permanent genetic defects that can give rise to cancer.[20,21] As an attestation to its importance, mutational inactivation of p53 is detected in close to half of all human cancers.[22] Accumulation of functional p53 leads to cell-cycle arrest, which allows for repair of damaged DNA before it can be propagated or elimination of cells with irreparable damage by apoptotic pathways.[15] Interestingly, the normal tanning response is dependent on the expression of p53,[23] and mice lacking p53 have a higher propensity to develop skin cancer in response to UVR.[24] Studies by Gilchrest and colleagues[16,25,26] have clearly demonstrated that tanning is a response to DNA damage. By introducing single-stranded DNA composed of the substrate for characteristic UVB mutations to both in vivo and in vitro models, tanning was induced in the absence of UVR. This response is p53-dependent and significantly less pronounced when alternate DNA sequences are used. Until recently, the mechanisms by which p53 was involved with this process were unknown. A landmark study by Cui and colleagues[22,27] surprisingly demonstrated that p53 itself is responsible for direct transcriptional activation of the proopiomelanocortin (POMC) gene, which encodes α-melanocyte–stimulating hormone (α-MSH), the ligand for the melanocortin-1 receptor (MC1R) found on the surface of melanocytes. These steps culminate in the intracellular accumulation of cAMP and melanin production (Figure 1). Collectively, these findings may explain why p53-deficient mice lack a tanning response and are less able to

protect themselves from harmful UVR and coincide with the finding of p53 mutations in over 90% of squamous cell carcinoma (SCC) and 50% of BCC.[28,29] Thus, p53 provides a critical role in the protection of human skin from the damaging effects of the sun. It should also be noted that several other keratinocyte-derived paracrine factors as well as melanocyte-derived autocrine cytokines contribute to the tanning response, and these processes are also p53-dependent.[30]

## The Future of Tanning Research

As each component of the tanning response is characterized, there becomes a target for the study of natural genetic variation and its relationship to cutaneous carcinogenesis. Polymorphisms in p53 and many of the downstream players in the pigmentation pathway are implicated in the differential propensity to develop skin cancer, particularly in those people with fair skin, blue eyes, and red hair.[31–33] Individual elements also become targets for drug development as a means of rescuing a defective or inefficient pigmentation pathway and will likely be the basis for the next generation of sunscreens and cancer prevention techniques. It is remarkable that application of single-stranded DNA can stimulate tanning in the absence of UVR. If any of the downstream constituents of the pathway shown in Figure 1 are ineffective, however, this response will be abrogated at the level of that defect. D'Orazio and coworkers[34] were able to demonstrate that application of a cyclic AMP derivative directly to the skin of mice lacking the MC1R was able to rescue the tanning response. Hence, normal tanning was induced in mice unable to tan in response to UVR, indicating the "availability" of the melanogenic machinery. Accordingly, in the presence of UVR, these mice had a longer duration of tumor-free survival compared to untreated mice.[34] This study and others that

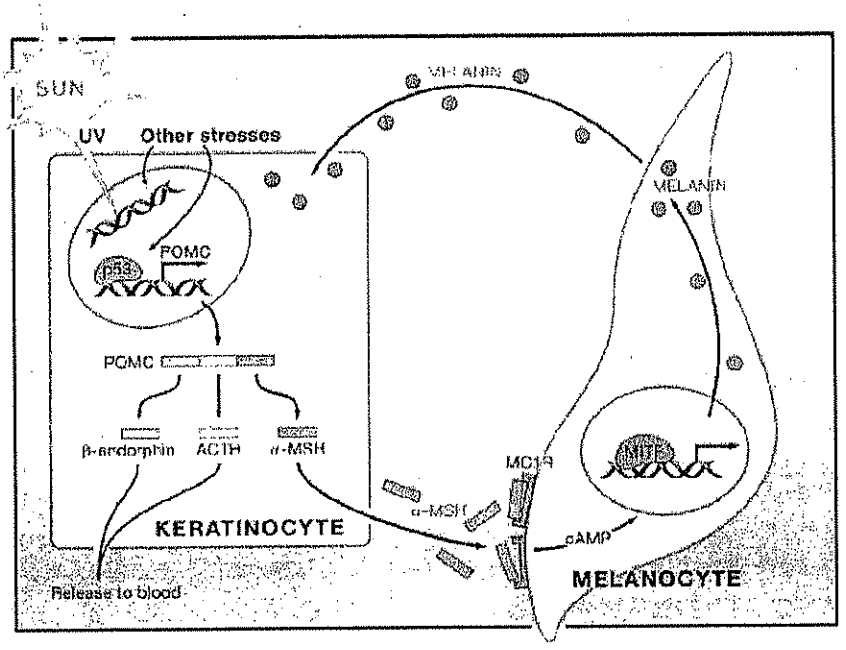


**Figure 1.** Pathway of UV-induced skin pigmentation (tanning). In response to UV-induced genotoxic stress, p53 becomes activated in skin keratinocytes and stimulates transcription from the *proopiomelanocortin* (*POMC*) gene promoter. The POMC precursor polypeptide is then processed into several bioactive products including α-MSH, which, through a paracrine effect on epidermal melanocytes (mediated by the α-MSH receptor MC1R and the melanocytic transcription factor MITF), leads to melanin production and redistribution among skin cells. The release of ACTH (adrenocorticotropic hormone) and the opioid peptide β-endorphin into the blood may relieve inflammation and contribute to sun-seeking behavior. (Reproduced, with permission, from Elsevier Press and originally appeared in Oren and Bartek).[22]

manipulate individual components of the tanning pathway[35–39] will have enormous impact on cutaneous research in determining whether such small-molecule topical therapies are possible in humans and whether they may provide protection against skin cancer. Patents have been issued for many of these approaches and specific compounds, and products will likely be entering trials in the near future.[40]

## UVR and Cutaneous Malignancy

There is little doubt that UVR is implicated in the development of skin cancer. The World Health Organization has estimated that in the year 2000, up to 71,000 deaths worldwide were attributable to excessive UV exposure.[41] In the United States, skin cancer accounts for more than 50% of all malignancies, with well over 1 million cases of nonmelanoma skin cancer[42–45] and 60,000 cases of malignant melanoma estimated for 2007 (melanoma in situ will account for an additional 46,000 cases).[46] Mortality from melanoma in the US occurs at the rate of approximately one life per hour or more than 8,000 deaths annually.[46] Although melanoma accounts for only 3% of total cutaneous malignancies, it is responsible for approximately 80% of skin cancer deaths.[47]

The rates of increase in both melanoma and nonmelanoma skin cancers are nothing short of alarming.[48] Skin cancer incidence is rising faster than that of any other cancer, with the lifetime risk for an American to develop melanoma estimated to have increased approximately 2000% in the past 75 years.[49] Among women under 40, the rate of BCC has tripled in the past 30 years, while that of SCC has quadrupled.[50] Melanoma is currently the second most common cancer after thyroid cancer among women in their 20s.[46] While many feel that this might be due to better surveillance and earlier detection, these factors alone cannot account for the observed changes.[49] UVR has been definitively shown to cause nonmelanoma skin cancer in both humans and animal models, but this link is more difficult to prove in the case of melanoma. Patients undergoing PUVA treatment develop a dose-dependent increase in SCC compared to controls,[51] as do rodents in well-designed studies examining UVR exposure.[52] Additional work in animal models of UVR-induced melanoma will continue to contribute to our knowledge of the disease process.[53] Melanoma, however, can occur in non–sun-exposed areas and is known to have both genetic and environmental components to its development. These become complex variables to study, because melanoma in several members of a family could represent a genetic susceptibility or common environmental exposures—or both. Despite these issues, it is generally accepted that few cases of melanoma can be attributable to germline mutations alone and that exposure to UVR remains the single most modifiable risk factor in the prevention of the disease. In support of this hypothesis, melanoma incidence varies by latitude and altitude, with those regions at higher altitude and closer to the equator (i.e., more UVR) having higher incidence of melanoma when corrections are made for baseline skin pigmentation.[54] Residents of Australia and New Zealand have the highest rates of melanoma in the world, because their citizens are largely fair-skinned and live close to the equator.[55] A meta-analysis by Elwood and Jopson[56] found a positive correlation between intermittent, intense sun exposure and malignant melanoma in 21 of 23 studies.[56] The rate of melanoma in Caucasian Americans is as much as 23 times higher than that for black Americans,[57] and among Caucasians, degree of pigmentation is a risk factor in virtually all epidemiologic studies, with a gradient of risk increasing from black to brown to red hair, and among those with light eyes.[58] In addition to these factors, the number of nevi, particularly those with atypical features, excessive sun exposure before the age of 18, and sunburn at any age have all been repeatedly demonstrated to increase one's lifetime risk for the development of melanoma.[59] Most recently, as we reveal more about the molecular mechanisms driving the tanning response, there is a growing cohort who feels that it is a person's ability to tan as opposed to baseline skin color that is a

better predictor for the risk of melanoma with exposure to UVR,[60] because this may represent an increased rate of DNA repair.[61] Overall, although the precise figures are difficult to ascertain, it has been estimated that 65% to 90% of melanomas are a result of UVR and that these numbers are sharply on the rise.[62] Yet given the growing body of evidence firmly linking UVR to the development of both melanoma and nonmelanoma skin cancer, the behavior of those at highest risk (i.e., fair-skinned individuals with a low ability to tan) has not changed accordingly.

## But Doesn't a Tan Protect Me from the Sun?

Among the largest misconceptions of those who tan from both natural and artificial sources is that a tan affords protection against further UV damage. Numerous studies have established that the photoprotective effect of an acute sun tan is equivalent to wearing a sunscreen with a sun protection factor (SPF) of 2 to 3.[63,64] To maintain this level of protection, however, one would have to receive ongoing exposure to UVR, which would be associated with other sun-related injuries beyond accumulation of DNA damage. Artificial tanning sources are predominantly UVA, with UVB comprising somewhere between 0.1 and 5% of emission depending on bulb specifications.[65–67] Because UVA is 2 to 3 orders of magnitude less efficient and has mechanistic differences from those changes caused by UVB, tanning beds provide even less protection, with an SPF between 1 and 2.[64,68] Thus, one of the most common reasons for people to tan—to protect against further damage from the sun, or the "prevacation" or "presummer" tan is a misconception. Any tan is an indication of DNA damage and potentially contributes to the development of cutaneous malignancy.[69] Moreover, among those with sun-seeking behavior, a tan from artificial or natural sources may encourage longer duration spent in the sun. Because there is minimal protection afforded by this tan, one is effectively increasing the risk for skin cancer by receiving higher amounts of cumulative exposure to UVR (Table 1).

**TABLE 1. Sun Protection Factors from Tanning**

| |
|---|
| SPF from tanning outdoors = 2–3 |
| SPF from tanning indoors = 1–2 |
| SPF from artificial tanning products = 2–3 (but often fortified with sunscreen) |

*These values only apply to acute changes, and lasting protection would require repeated exposure to UVR or reapplication of artificial tanning products. Because many believe that these figures are much higher, they may be spending more time in the sun and at tanning salons, thus putting themselves at even greater risk for skin cancer.[63–64,68,129–131]

## Tan Now, Worry Later

While the deleterious manifestations of sun exposure and excessive tanning are not seen for many years, the perceived personal and social value of tanning is immediately apparent. As such, it is difficult to prove convincingly that factors in one's youth are responsible for disease 30 to 50 years later, yet epidemiologic data of who tans and why they tan are currently quite robust. There is strong evidence to support the fact that sun exposure during childhood, specifically intermittent sun exposure, is linked to the development of skin cancer in adulthood.[70,71] Children are believed to receive up to 3 times more UVB than adults because of midday sun exposure during the summer,[72] and it is believed that those who receive high levels of sun as a child carry this behavior in to adulthood.[50] Dermatologists frequently inform their patients that up to 80% of lifetime UV exposure occurs before the age of 18 based on a study by Stern and colleagues[73] that estimated regular use of sunscreen during the first 18 years of life would reduce the incidence of nonmelanoma skin cancers by 78%. Recent studies by Godar and colleagues,[74] however, indicate that this may not be the case. While sunscreen use in childhood may very well reduce the incidence of skin cancer later in life, these reports state that Americans get less than 25% of their lifetime UV dose by 18 and that it is older men who get the most UV exposure. In fact, advanced age and male gender carry a significantly higher risk of death from melanoma compared to other groups.[75] In any case, early educational intervention and lifelong prevention will likely be key factors in slowing the rates of skin

cancer and associated morbidity. Surprisingly, as further evidence points toward cumulative dose of UV exposure over one's lifetime being the critical parameter in the development of cutaneous malignancy, there has been essentially no modification in the behavior of tanners. A 1998 survey of more than 1,000 American teens aged 11 to 18 revealed that 72% had been sunburned the previous summer.[44] When the study was repeated in 2004, this figure was unchanged.[44] Rates of sunscreen use have decreased since 1996,[76] while tanning salon use has increased dramatically. In a study of more than 10,000 adolescent Americans, girls were more likely to use sunscreen than boys (34% reported use overall), but were also likely to have received at least three sunburns the previous summer and to state that it was worth burning to get a tan.[72] These statistics are remarkable when one considers that a single severe sunburn in childhood may increase the risk of melanoma twofold.[77]

## The Double Hit

The indoor tanning industry has been the subject of intriguing literature and controversy in the dermatologic community.[78,79] Although there are conflicting opinions on specific aspects and associations, it is clear that those who tan outdoors are much more likely to frequent indoor tanning facilities, thus multiplying their exposure to UVR.[80] The growth of the tanning industry worldwide is staggering. In the United States, tanning generates $5 billion annually, up from $1 billion in 1992.[81] On average, 1 million people tan daily, and 70% are Caucasian women aged 16 to 49.[81–83] The prevalence of tanning among teenagers varies among studies and geographic location. Lazovich and Forster[84] have pooled this data and indicate that approximately 30% of teens aged 13 to 19 are tanning, with girls being two to three more times likely to tan indoors than boys. Adolescents who use sunscreen or other means of sun protection are much less likely to be indoor tanners.[84] Surprisingly, tanning bed use is no different among those with either a maternal diagnosis of skin cancer or a family history of melanoma compared with nonaffected adolescents.[85] Among adolescents who tan, 77% planned to continue doing so, while 22% of those who did not tan planned to start. Indoor tanners are less likely to use sun protection, are less knowledgeable about skin cancer risks, are more likely to agree that tans are attractive, and are more strongly influenced by social factors compared with nontanners.[86] Frequent tanning bed use in teenagers has been associated with being highly concerned about weight, frequent dieting, using laxatives or vomiting to control weight, smoking cigarettes, binge drinking, and use of recreational drugs.[87] Thus, there is an association between tanning and a range of health risk behaviors and social influences (Table 2).

## Tanning Bed Use and Skin Cancer

Emerging evidence is making it difficult to dispute the link between tanning bed use and the development of both melanoma and nonmelanoma skin cancers. This is of no surprise, because the molecular alterations in the skin of artificial tanners are the same as in the skin of those who tan from the sun.[88] As mentioned, the proportion of UVB from artificial sources ranges from 0.1% to 5%,[65–67,89] and the effective incident energy of light varies as a function of these differences, sunbed design, distance to the skin, power, bulb age, and of course, duration of tanning. Average tanning bed output in one study was measured as 192 $W/m^2$ UVA and 0.35 $W/m^2$ UVB.[90] Total doses of UVR from a tanning bed may be as much as five times than those from natural sunlight, such that 20 minutes spent at a tanning

**TABLE 2. Characteristics of Indoor Tanners**

| |
|---|
| More likely to tan outdoors |
| Less likely to use sun protection |
| Less knowledgeable about skin cancer risks |
| More influenced by social factors |
| More concerned about weight |
| More likely to smoke |
| More likely to binge drink |
| More likely to use recreational drugs |
| More likely to have parents who tan |

salon may be equal to 2 to 3 hours in the noontime sun.[91,92] Again, this is in addition to the UVR received from natural sunlight while tanning outdoors. This high-intensity repeated exposure to UVR is a new phenomenon previously not experienced by humans, and its effects on cutaneous carcinogenesis are becoming evident.

Numerous studies have investigated the association between tanning bed use and the development of skin cancer, as well as more recent meta-analyses that pool these results.[91–95] Initial studies presented inconclusive views as to whether indoor tanning increases the risk for cutaneous carcinogenesis and, in particular, malignant melanoma. More statistically powerful, better designed studies have since emerged and have essentially confirmed the link. Many of the early publications were small, case-control studies. These reports were based on a patient's memory of distant events and, hence, subject to recall bias, particularly in those patients who already carried a diagnosis of melanoma that were asked to remember past sun exposure.[96,97] Beyond recall bias, limitations in interpreting studies on the association between indoor tanning and skin cancer include the difficulty to correct for sun exposure from ambient light and outdoor tanning and the nature of the emission from different tanning lamps across tanning centers. Furthermore, lamps in use before the 1980s had significantly higher levels of UVB emission.[65,98] Finally, many studies compared "ever" versus "never" populations of patients, so depending on study design, those subjects who reported a single use of a tanning bed may be included in the same cohort as those who tanned multiple times per month for many years.

The study by Veierød and colleagues[99] is perhaps the best designed to date. This investigation prospectively studied 106,379 Scandinavian women for an average of 8 years to determine factors that may be associated with an increased risk of melanoma. The results demonstrated a statistically significant 55% increase in risk for young women with a history of 40 hours or more of sunbed use after adjustment for sun sensitivity and measures of sun exposure. In this study, data were collected prior to the diagnosis of melanoma, so it was less prone to recall and selection biases. Westerdahl and coworkers[65] reported a dose-dependent risk for melanoma with regular use of tanning beds after adjustment for hair color, raised nevi, skin type, and number of sunburns. This risk was more pronounced in indoor tanners under age 36. The most recent and in-depth meta-analysis performed by The International Agency for Research on Cancer found several positive associations between indoor tanning and melanoma, particularly with exposure before age 35. They were also able to demonstrate an association with SCC.[91] Other studies have linked BCC with indoor tanning, including one study that reported nearly double the number of lifetime visits to a tanning salon among young women with proven BCC.[100,101] Diffey estimated the mortality from melanoma related to UVA sunbed use in the United Kingdom to be 100 lives annually.[102] Taken collectively, these data strongly suggest that the link between indoor tanning and the development of cutaneous malignancy is substantial and responsible for significant, yet entirely preventable, morbidity and mortality.

## Tanning, the Government, and Powerful Lobbying

As the link between tanning and skin cancer becomes apparent, there has been a move to introduce legislation to regulate the tanning industry. The Food and Drug Administration (FDA) and the Federal Trade Commission (FTC) specify equipment performance standards, require protective eyewear to be available to users, and prohibit deceptive advertising purporting the health benefits of indoor tanning.[103] At present, 25 states have age restrictions on tanning—some have an absolute age restriction (ranging from 13 to 16), while others require a parent to either be present or write a note of consent.[104] At the majority of facilities, these notes are valid for 1 year. There are currently many introduced legislations that can be found at the National Conference of State Legislatures.[104,105] Restrictions on indoor tanning are an

important first step in reducing the damage that eventually leads to skin cancer, yet enforcement of these laws and penalty for their violation is effectively nonexistent. Studies in the United States and abroad indicate that up to 88% of tanning businesses illegally allowed minors to tan without parental consent.[66,106,107] In one study 90% of centers allowed clients with Type I skin to tan and 75% of these facilities provided reassurance of the safety of tanning to Type I customers.[107] Sixteen percent of Texas facilities charged for eyewear despite state regulation mandating that it be provided free of charge[108] (although this may be insignificant because up to 40% of teenagers admit to not using eyewear at all[86,109]). Approximately 75% of indoor tanning advertisements promote unlimited tanning, 95% allow customers with these packages to tan as frequently as desired, and 100% offer unlimited tanning packages.[110] Unfortunately, this advertising is becoming increasingly marketed to adolescents via high school newspapers.[111] Sixteen percent of frequent tanners had more than 100 sessions per year, and recommended exposure limits as stated by the FDA were exceeded by 95% of patrons.[90] To conclude that current regulations are ineffective is an understatement, and a visit to the Web site of the International Tanning Association (ITA) may give an indication as to why this is the case.[11] On the front page, one can read in large uppercase font, "Help defeat the California under 18 ban," and "Help defeat the Massachusetts teen tan ban," and it becomes immediately apparent that big money equals powerful lobbying and a veritable blockade on Washington. The ITA touts the benefits of controlled tanning and "government-regulated controls" in place to "ensure safety, consistency, and optimal exposure (unlike the outdoors)" and that vitamin D "wards off a host of debilitating and sometimes deadly diseases."[11]

### The Vitamin D Debate

A large body of literature has provided the tanning industry with ammunition in the ongoing battle to restrict access to their facilities. These studies alluding to health benefits from tanning span a variety of conditions from decreased incidence of internal malignancies and decreased rates of bone fractures, diabetes, multiple sclerosis, hypertension, systemic lupus erythematosus, and numerous other conditions.[81,95] The majority of these reports focus on the role of vitamin D in normal physiology such as calcium homeostasis and maintenance of proper immune function, and many incorrectly imply that intentional, unprotected exposure to the sun or tanning bed usage is necessary to maintain adequate serum levels.[112–114] One frequently cited study supporting an essential role for the photosynthesis of vitamin D[115] was later shown to be supported by the UV Foundation, a staunch supporter of the commercial tanning industry, and to contain many flaws in study design.[116] A comprehensive review by Wolpowitz and Gilchrest[112] addresses many of the individual studies supporting UVR exposure and purported health benefits. Many of these conclusions are quite controversial, stem from poorly designed studies, are observational in nature, or involve toxic levels of vitamin D in in-vitro studies or animal models.[112] Furthermore, what they largely fail to address is that although vitamin D does indeed provide a critical role in numerous cellular processes, the amount of UVR needed to produce sufficient levels of vitamin D is small and does not by any means justify the use of artificial tanning sources or offset the risk of cutaneous malignancy associated with their use.[81] Maximal cutaneous production of vitamin $D_3$ occurs in less than 5 minutes at noon in June in Boston, Massachusetts.[112,117] Greer[118] demonstrated that fair-skinned, clothed infants required only 0.5 to 2 hours of sun exposure per week to maintain adequate levels of vitamin D. Studies examining vitamin D levels in individuals who used sunscreen daily versus controls reveal that none of the participants had levels below the normal range.[119] Maximal vitamin D photosynthesis occurs at suberythemal doses of UVR in all individuals and longer exposure has no effect on vitamin D levels while UVR-induced DNA damage increases linearly with continued exposure.[120] Better-designed epidemiologic studies have failed to substantiate a

role for vitamin D in the prevention of cancer or other systemic diseases,[121,122] and no clinical trials have confirmed a link between higher levels of vitamin D and autoimmune disease.[112] Moreover, if any benefits of increased UVR were proven robustly, these results would also be achieved entirely by dietary supplementation with vitamin D. Daily intake of two 8-oz glasses of fortified milk or orange juice, one standard vitamin tablet, or incidental exposure of the face and backs of the hands to 0.25% minimum erythema dose of UVB three times per week is sufficient to achieve normal serum levels of vitamin D.[112] Thus, advocating exposure to a known carcinogen while a completely safe alternative exists is an extremely negligent suggestion, particularly when one in three Caucasians will develop skin cancer.

## Is It Possible to Be Addicted to Tanning?

Complicating matters are recent reports implicating frequent tanning as a type of substance-related disorder.[123] Fifty-three percent of frequent tanners in one study met the official criteria for a substance-related disorder as defined by the American Psychiatric Association.[124] Feldman and colleagues[125] showed that UVR is a reinforcing stimulus in frequent indoor tanners by demonstrating their ability to distinguish between otherwise identical UV and non-UV tanning beds in 95% of cases. Revisiting the tanning pathway in Figure 1 reveals details that may partially explain the psychology of tanning. Every dermatologist has certainly heard patients state that they are "addicted to the sun." There may be a scientific basis for such statements if one examines the by-products of tanning. POMC is a precursor protein that is proteolytically cleaved to numerous smaller peptides that are released into the blood, including β-endorphin (Figure 1). Additionally, there is evidence that melanocortins regulate sexual function.[126] Thus, a source for a legitimate "tanner's high" certainly warrants further investigation. To this end, one small study was able to exhibit opioid withdrawal symptoms in 50% of frequent tanners who underwent chemical opioid blockade.[127]

## So Where Do We Go from Here?

The link between UVR from natural and artificial sources and skin cancer is becoming much like the link between cigarettes and lung cancer or alcohol consumption and cirrhosis of the liver. In those cases, national age legislation, severe penalties for their violation, and enormous public awareness campaigns have made tremendous strides in reducing teenage abuse of these substances. It is clear that tanning is now at a similar crossroads. To date, minimal progress has been made in improving sun protection practices and reducing sun exposure, particularly among adolescents despite their knowledge of the links to skin cancer. It is unreasonable to prohibit anyone with blue eyes or red hair from using tanning facilities, and public awareness of the risks of tanning has done little to change behavior. Table 3 lists some potential interventions in the current state of tanning that may be more effective in implementing change. At present, the WHO is suggesting a complete ban on indoor tanning for anyone under the age of 18.[41] In Sweden, where sun protection measures, melanoma surveillance campaigns, and changes in tanning habits are more actively encouraged, the increased rates of melanoma appear to be slowing.[128] In the United States, The Senate has recently overwhelmingly passed the Tanning Accountability and Notification Act (TAN) in an effort

**TABLE 3. Suggested Interventions to Decrease the Use of Indoor Tanning Facilities**

| |
|---|
| Absolute age limits (<18 years) |
| Improved measures to enforce parental consent |
| Stronger enforcement of current regulations with steep penalties for violations |
| Early education as to the deleterious effects of tanning (i.e. preschool) |
| No advertising to minors |
| No deceptive advertising |
| Surgeon General's warning on all artificial tanning sources |
| Taxation |
| Reduction of UV dose per session (leading to the same cosmetic results) |
| Encouragement of primary care provider interaction |
| Home tanning bed regulation |
| Large-scale public education campaigns |

to raise public awareness regarding the hazards of tanning beds. Sunless tanning booths are increasing in popularity and provide a safer alternative to UV-induced tanning.[129–131] These centers use misters to apply an even coat of dihydroxyacetone (DHA), the only color additive approved by the FDA as a tanning agent. It should be reinforced, however, that they provide negligible sun protection.[130] Consistent with a higher "tan-promoting attitude," users of DHA are largely unaware of this fact and are more likely to have recently received a sunburn or used a tanning bed than those who do not use artificial tanning products.[130] Many manufacturers of sunless tanning agents are incorporating sunscreens to address the misconception of a protective role associated with their use.[131] Early intervention will certainly be a key factor in establishing good practice sun protection from a young age, including covered recreation areas at schools, incorporation of skin examinations at well-child visits, and reinforcement from parents and primary care physicians.[132]

## Conclusions

Exposure to UVR provides humans with a sense of well-being and results in a physical change in the appearance of their skin known as tanning. It is now evident that although these changes are perceived as desirable, tanning represents a response to DNA damage from UVR, and that this exposure is strongly implicated in the development of cutaneous malignancy. As the public becomes increasingly aware of this link, there has been little in the way of behavior modification despite an exponential surge in the incidence and mortality from skin cancer. There is a misconception that tanning provides protection from further UVR and this false notion is perpetuated by the indoor tanning industry. Thus, frequenters of indoor tanning facilities are less likely to use sun protection and are more likely to tan outdoors, greatly multiplying their exposure to UVR and increasing their risk for skin cancer. Basic research in tanning will lead to the development of small-molecule activators of the pigmentation pathway that may provide higher levels of protection than current sunscreens. Until these products are commercially available, further changes in public awareness regarding the effects of UVR and in the legislation regulating the tanning industry must be made and enforced to reduce the burden of skin cancer on society.

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